Miklos and Saye assumed a general success rate of 85% for the Burch colposuspension. However, I take the liberty of remembering that none of our patients had undergone previous surgery and that those with urethral sphincter weakness or detrusor instability were excluded. This means that our population represents selected patients with primary uncomplicated pure genuine stress incontinence. Moreover, subjects were followed up for a relatively brief period. I believe that in such circumstances a success rate around 95% should be presupposed.[2, 3]

We began our trial with the conviction that the paravaginal repair would have been an excellent antiincontinence operation. I now discourage its use in treating stress incontinence. Nonetheless, I do not believe that it should be excluded from our surgical armamentarium. At our institution we are continuing to perform the paravaginal repair but exclusively in continent patients (or those with only minimal degrees of incontinence) with a cystocele from a lateral defect who are undergoing abdominal surgery for other reasons.

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6/8/78533

Efficacy of methotrexate

To the Editors: I was pleased to read the article by Stika et al. (Stika CS, Anderson L, Frederikson MC. Single-dose methotrexate for the treatment of ectopic pregnancy: Northwestern Memorial Hospital three-year experience. Am J Obstet Gynecol 1996;174:1840-8.) The results of Stika et al. showing only a 64% success rate of resolution of ectopic pregnancy with a single methotrexate injection contrasts with the 85% to 95% success rates of other authors.

In fact, there are few reports showing limited success with methotrexate. In our study[1] 50 mg of methotrexate was injected directly into a laparoscopically confirmed ectopic pregnancy. Of 44 patients treated this way, only 27 (61.4%) had successful resolution and 17 (38.6%) required a further procedure, usually salpingostomy or salpingectomy.

Treatment for ectopic pregnancy by methotrexate requires the patient to be under prolonged observation, with failure occurring up to several weeks after the injection, even in the face of decreasing β-human chorionic gonadotropin levels. These patients must be carefully selected and counseled. It is probable that when other centers review their results with single-dose methotrexate, they will find a success rate below the classically reported 97% reported by Stovall and Ling.[2]

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6/8/78615

Endoscopic coverage of fetal open myelomeningocele in utero

To the Editors: Harrison was mistaken when he stated that endoscopic coverage of myelomeningocele has not yet been attempted in human fetuses (Harrison MR. Fetal surgery. Am J Obstet Gynecol 1996;174:1255-64). In fact, we have successfully performed an experimental procedure designed to prevent ongoing exposure of the spinal cord to amniotic fluid in two patients at Vanderbilt University Medical Center. This minimally invasive fetal surgery was developed by us in pregnant mixed-breed ewes.[1] The technique we designed consists of placement of a maternal split-thickness skin graft over the exposed neural placode. The skin graft and a covering of oxidized regenerated cellulose (Surgicel, Johnson & Johnson Medical, Arlington, Tex.) are attached in a carbon dioxide atmosphere with fibrin glue prepared from autologous cryoprecipitate.

Two fetuses with open lumbar myelomeningocele underwent endoscopic coverage of the spinal lesion at 22 and 23 weeks' gestation. One infant, delivered by planned cesarean section at 35 weeks' gestation after demonstration of fetal lung maturity, is 1 year old. The other fetus was delivered 1 week after operation because of the development of amnionitis and died in the delivery room of extreme prematurity.[2]

A growing body of evidence suggests that, in addition to the congenital neurologic defects associated with the development of myelomeningocele, spinal cord injury may result from prolonged exposure of the neural elements to amniotic fluid.[3] In several animal models the clinical and pathologic manifestations of surgically induced "myelomeningocele" were prevented by in utero repair.[1] We are currently conducting in vitro research in an attempt to identify the timing and etiologic agents of the environmental insult more precisely. In the meantime we agree that surgical treatment of this nonlethal defect is not justified with a standard hysterotomy approach, because of the unacceptably high morbidity and mortality associated with open fetal surgery. Minimally

invasive fetal surgery, however, appears to constitute a feasible approach to myelomeningocele and other nonlethal malformations that result in progressive organ damage.

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6/8/78530

Reply

To the Editors: I thank Bruner et al. for updating the evidence that there is ongoing neurologic injury to the spinal cord in fetuses with myelomeningocele. There is also convincing evidence from our fetal lamb model that exposing the normal lumbar spinal cord directly to the amniotic cavity for the second half of gestation produces a human-like myelomeningocele at birth[1] and that covering such an experimental myelomeningocele lesion early in gestation prevents neurologic damage.[2] More recently, we studied therapeutically aborted human fetuses with myelomeningocele and have demonstrated evidence for traumatic damage to the exposed neural tissue acquired in utero and during delivery.[3] Also we studied human fetuses with myelomeningocele at autopsy and demonstrated that the musculus latissimus dorsi flap technique developed in fetal lambs is applicable to human fetuses.[4] However, we remain reluctant to apply this clinically until some important unanswered questions about the natural history of human fetal myelomeningocele are solved. Most important, does the fetus with a myelomeningocele move the lower extremities actively early in gestation and lose that ability later in gestation? In other words, is there neurologic function to salvage by in utero repair? Although fetal leg movement is easy to see by sonogram, it has proven very difficult to distinguish active from passive or reflex-mediated movement of the lower extremities.

From our extensive experimental work we remain unconvinced that a skin graft from any source, especially one attached only with glue, will protect the spinal cord and affect outcome. We believe this will require a composite muscle flap, which can be accomplished either by open fetal surgery or, more recently, by FETENDO (fetal endoscopic surgery) techniques. From available information about the two patients in Tennessee, we remain unconvinced that the procedure affected outcome in the fetus who survived (it is not clear that the skin graft stayed). It is also unclear that the endoscopic approach used in these two fetuses is safer than fetal surgery because uncontrollable preterm labor led to delivery and death 1 week after the procedure in one of the two cases. Nevertheless, this remains an exciting area for further research and careful clinical application in the future.

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6/8/78531

Congenital diaphragmatic hernia: Can prenatal ultrasonography predict outcome?

To the Editors: We read with interest the article of Dommergues et al. (Dommergues M, Louis-Sylvestre C, Mandelbrot L, Oury JF, Herlicoviez M, Body G, et al. Congenital diaphragmatic hernia: can prenatal ultrasonography predict outcome? Am J Obstet Gynecol 1996;174:1377-81) on congenital diaphragmatic hernia and we would like to confirm their data. Ultrasonographic prenatal diagnosis of congenital diaphragmatic hernia is well established, but the correlation of prenatal detection with clinical outcome remains unclear.

In a 5-year period in the I. Department of Obstetrics and Gynecology, Semmelweis University Medical School, Budapest, we designed a retrospective study of 26 patients with congenital diaphragmatic hernia (21 left, 2 right, 2 bilateral, 1 anterior) diagnosed prenatally. In isolated left congenital diaphragmatic hernia we have found the ultrasonographic triad of potential prenatal prognostic factors in all the patients diagnosed at <25 weeks' gestation: polyhydramnios, intrathoracic stomach, and major mediastinal shift.[1] The small abdomen was found in only 3 patients. None of the 10 fetuses or infants who had multiple malformations survived. Of the 16 cases of isolated congenital diaphragmatic hernia,